

a variety of antiseptic dressings were applied without appreciable change. It was then decided to consider the question of a sympathectomy. The skin of the foot showed four degrees of a rise during spinal anaesthesia. As the temperature of the theatre was seventy degrees Fahrenheit, this rise was taken to indicate a degree of vasospasm justifying operative interference. Accordingly on the 11th November, 1933, with the help of Mr. H. P. Malcolm, the right lumbar sympathetic chain was exposed by displacing the caecum and ascending colon towards the left. The second, third, and fourth sympathetic ganglia and their connecting strands were excised. The post-operative response was rapid and striking. Pain disappeared almost immediately, and within a few days the sloughs were thrown off and healing commenced. The ulcer was healed before the patient was allowed out of bed, three weeks after operation. Twelve months later the ulcer is represented by a small scar under the nail. The temperature of the legs has been noted periodically since. Typical figures are :—

14th January, 1934 - Room temperature 60°F., right leg 91°F., left leg 65°F.
 5th April, 1934 - - - Room temperature 68°F., right leg 92°F., left leg 82°F.
 17th September, 1934 - Room temperature 68°F., right leg 92°F., left leg 90°F.

PATHOLOGICAL REPORT.

Dr. P. A. Clearkin kindly examined the ganglia removed at operation. His report reads: "Many of the ganglion cells were degenerate, their outline distorted, cytoplasm badly stained, nucleus eccentric or missing. There was an increase in the amount of fibrous tissue, much of which appeared to be of recent formation. Though there was no evidence of acute inflammatory process in the sections examined, the number of polymorphonuclear leucocytes present in the lumen of the capillaries was larger than that usually seen in sections."

My best thanks are due to Professor P. T. Crymble for permission to publish the report of this case.

A Primary Tumour (Mixed-Cell Sarcoma) of the Pleura

By **S. B. BOYD CAMPBELL, M.D., F.R.C.P.ED.,**

AND J. S. YOUNG, M.A., M.D., B.SC.,

from the Royal Victoria Hospital, Belfast

A SCHOOL teacher, aged 44, was first examined on 11th October, 1933. Apart from an operation for appendicitis eight years previously, she had had no serious illnesses. She gave a history of sudden onset of pain in right side of chest one year ago, which lasted a week and was worse on breathing or on movement. Since then she has complained of slight soreness radiating towards the right shoulder, and of progressive weakness. Two weeks prior to examination she began to suffer from dyspnoea, which was worse on exertion. There was no oedema of her ankles, and her bowels and kidneys were functioning normally.

On examination her general condition was good, apart from some cyanosis. There was marked dullness of the whole of the right side of the chest up to the level of the third rib, with complete loss of breath sounds. X-ray showed that this dullness was due to a large effusion. On aspiration, two pints of bloodstained fluid were removed, and a similar quantity on 6th November, 1933. The radiogram taken immediately after the removal of the fluid showed a thickening of the basal pleura (fig. 1). As the effusion rapidly reappeared she was admitted to the Royal Victoria Hospital at the end of December, and an artificial pneumothorax after the removal of the fluid showed, on X-ray examination, multiple round shadows in the area of the collapsed lung and protruding from the peripheral pleura (fig. 2). A clinical diagnosis of pleural endothelioma was made. The rapid recurrence of the fluid necessitated frequent aspiration of one to two or more pints of bloodstained fluid, the patient only obtaining relief from her dyspnoea and discomfort after each aspiration.

The patient lived for five months after the last radiological examination (22nd January, 1934). During this period, the tumour involving the right pleural sac had grown rapidly. At the post-mortem examination on 6th June, 1934, the right lung was collapsed and was ensheathed by a mass of cellular tumour tissue which measured three inches in thickness over the costal surface of the lung and filled the pleural sac. The tumour was necrotic and extremely friable, but its natural relations were preserved as far as possible by stripping the parietal pleura from the chest wall. No growths, either primary or secondary, were observed in any part of the body outside the chest, and the breasts were normal. Both bronchi were congested, but neither the right nor the left showed any evidence of ulceration. In view of these findings a provisional diagnosis of "endothelioma of the pleura" was made at the post-mortem examination. The appearances of a coronal section through the fixed specimen are illustrated (fig. 3). Two small nodules of growth are present in the substance of the lung: they occupy a superficial situation in direct continuity with the pleural mass. The bronchial lymph-glands are anthracotic, but they are not notably enlarged and they show no evidence of invasion by the growth. Microscopically, the growth is extremely cellular and shows very extensive necrosis. In general, the tumour cells are small and spheroidal or ovoidal, but perhaps the most conspicuous feature is the large number of multinucleated giant cells (fig. 4). No papillary proliferation is apparent, and it is only occasionally that an alveolar structure is suggested. There is an abundant formation of reticulum. In short, the tumour is best described as a mixed-cell sarcoma.

Diffuse pleural growths which form a thick investment of one lung are uncommon, nevertheless the present case was the third of a series of three (Ser. Nos. A1014, A1026, A1113) observed within five months. In all three cases the growth was confined to one pleural sac, with or without gross involvement of the bronchial lymph-glands on the same side. In one case (A1014) a simple tumour, namely, a cortical adenoma, was found in the left adrenal, but no malignant growth, either primary or secondary, was disclosed by a thorough post-mortem examination in any other part of the body, either in this case or in the other (A1026). The bronchi were free from

ulceration in both cases. Microscopically, these two pleural tumours possess a papillary or an alveolar structure, and are composed of spheroidal cells with vesicular nuclei; they contain no reticulum. They have many features in common, but they differ in the respect that giant multinucleated cells are numerous in A1026 and absent in A1014.

The classification of pleural tumours of this type is uncertain. It has not even been established that they are primary growths of the pleura. Indeed, their primary nature has been vigorously contested by Robertson and by Willis and others. These authors are of the opinion that, with the exception of rare primary sarcomata of the pleural tissues, all such growths are "secondary, representing extensions, implantations, or metastases from an unrecognized or latent primary source, usually the lungs." They base their opinion principally on the diversity of the histological picture of "endotheliomata of the serous membranes," and assume too readily perhaps that the structure or the cytology of the serosal endothelium lining the pleural sac is quite inflexible. I agree with these authors that the term "endothelioma of the serous membranes" is a misnomer. Ten years ago I had an opportunity to study two more tumours of this pattern, and one of them proved to be a squamous epithelioma on microscopical examination. At that time I was inclined to think that Robertson's explanation was correct, and that the squamous epithelioma of the pleura had been secondary to a latent primary source, which had been overlooked at the post-mortem examination. A few years later, however, I was able to show that the serosal endothelium covering the visceral pleura of the rabbit can undergo metaplasia to an epithelium of cubical, columnar, transitional, or stratified squamous type. Since such a wide range of metaplasia can be induced experimentally, it can scarcely be denied that it may occur under natural conditions even in the human subject. If a malignant growth were to supervene upon any such metaplasia—and this is a common enough sequence of events in other situations—the histology of the growth must be as diverse as the metaplasia. Every pathologist is aware that a primary growth may be small or even insignificant compared with the bulk of its secondary growths, but it is hard to believe that pleural tumours, which are characterized above all things by their bulk and by their habit of ensheathing the lung, are generally secondary to a latent primary source in the lung, whereas gross primary sources in the same organ are seldom attended by any considerable infiltration of the pleura. In view of these considerations I cannot accept the generalization that all diffuse tumours of the pleura are secondary to a latent primary source in the lung, and, as a matter of fact, I hold that the four tumours mentioned in this short report are *primary* tumours of the pleural serous membrane, in spite of the diversity of their microscopical appearances.

REFERENCES.

-
- ROBERTSON, H. E., *Journ. Cancer Research*, 1924, Vol. 8, p. 317.
WILLIS, R. A., "The Spread of Tumours in the Human Body," 1934, J. & A. Churchill.
YOUNG, J. S., *Journ. Path. and Bact.*, 1928, Vol. 31, p. 265.

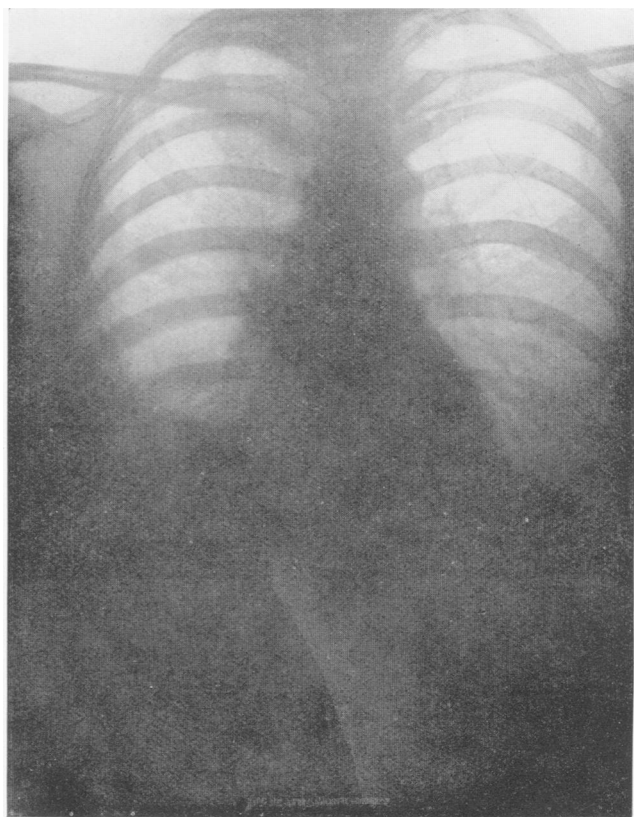


FIG. 1.

6 11 1933.—Radiogram immediately following removal of two pints of fluid from right side of chest, showing thickening of basal pleura.

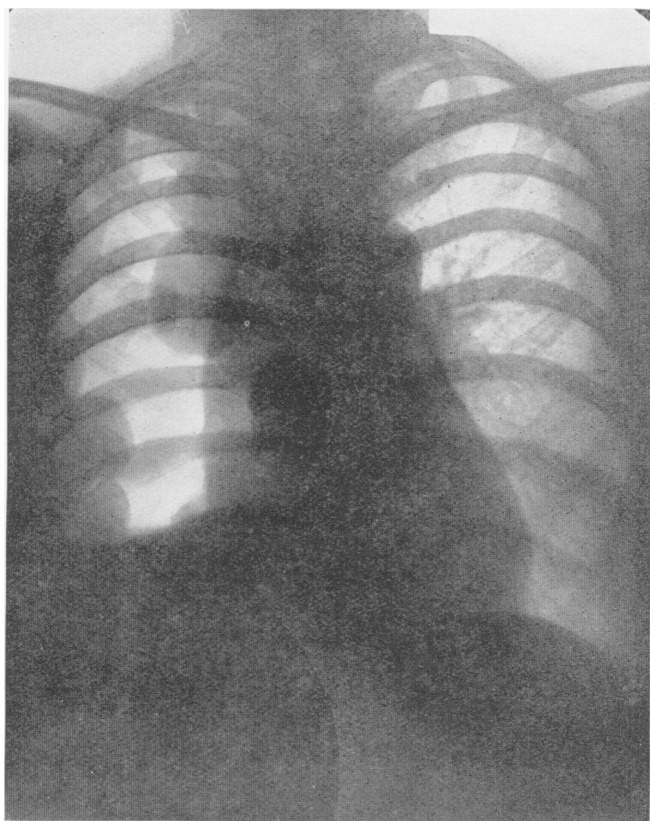


FIG. 2.

3 1 1934.—Radiogram following removal of fluid and replacement by air, showing large nodular growths in pleura.

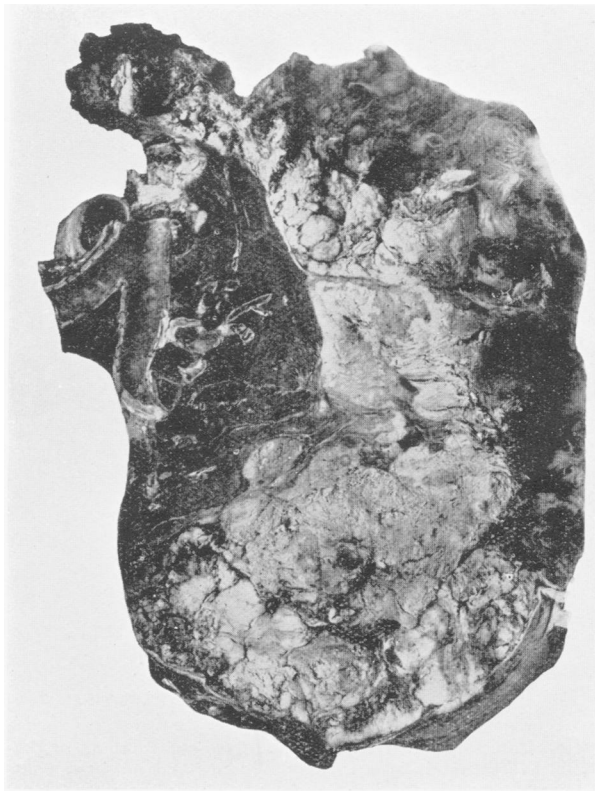


FIG. 3.

Coronal section through fixed specimen of primary mixed-cell sarcoma of the pleura.

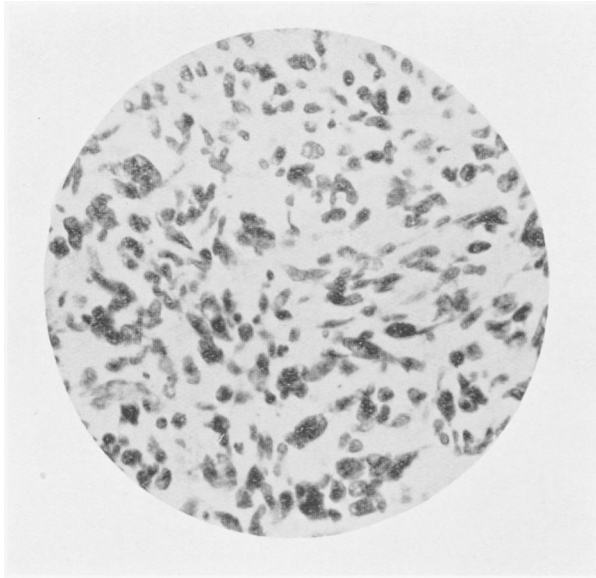


FIG. 4.

Microscopic section from primary mixed-cell sarcoma of the pleura. Note that it is extremely cellular, and note the conspicuous feature is the large number of multinucleated giant cells.